nary. On the other hand, the high number of species found during the present study suggests that trematodes represent the dominant component of helminth fauna of freshwater fish of the Peninsula, similar to observations of Scholz et al. (1995a, b) and Salgado-Maldonado et al. (1997).

The authors are indebted to Raúl Simá-Alvarez, Clara Vivas-Rodríguez, and Edgar Mendoza-Franco (CINVESTAV-IPN, Mérida, México) for help in collecting and examining fish, and to Eva Matějovská (formerly Faculty of Biology, University of South Bohemia, České Budějovice) for help in evaluation of the material. Thanks are also due to Martina Borovková (Institute of Parasitology, České Budějovice) for help in staining and mounting voucher specimens. This study was supported in part by the grant PO99 of the Comisión Nacional para el Conocimiento y Uso de la Biodiversidad (CONABIO), Mexico.

Literature Cited

Jiménez-Guzmán, F. 1973. Tremátodos digéneos de peces dulceacuícolas de Nuevo León, México I. Dos nuevas especies y un registro nuevo en el carácido *Astyanax fasciatus mexicanus* (Filippi). Cuadernos del Instituto de Investigaciones Científicos, Universidad Autónoma de Nuevo León, México, 17:1–19.

Kloss, G. R. 1966. Helmintos parásitos de espécies simpátricas de *Astyanax* (Pisces, Characidae). I. Papéis avulsos do Departamento de Zoología, Secretaría de Agricultura, São Paulo, Brasil, 18(17): 189–219.

Lamothe-Argumedo, R., and G. Ponciano-Rodríguez. 1986. Revisión de la subfamilia Acanthostominae Nicoll, 1914. Anales del Instituto de Biología, Universidad National Autónoma de México, Seria Zoología, 56:301–322.

Moravec, F., C. Vivas-Rodríguez, T. Scholz, J. Vargas-Vázquez, E. Mendoza-Franco, and D. González-Solís. 1995a. Nematodes of fishes from cenotes (sinkholes) in the Yucatán, Mexico. Adults. Folia Parasitologica 42:115–129.

———, ———, ———, ———, ———, **J. J. Schmitter-Soto, and** ———. 1995b. Nematodes of fishes from cenotes (sinkholes) in the Yucatán, Mexico. Larvae. Folia Parasitologica 42:199–210.

Pérez-Ponce de León, G., L. García-Prieto, D. Osorio-Sarabia, and V. León-Regagnon. 1996. Listados faunísticos de Mexico. VI. Helmintos parásitos de peces de aguas continentales de México. Universidad National Autónoma de México, Instituto de Biología. 100 pp.

Salgado-Maldonado, G., R. Pineda-López, V. M. Vidal-Martínez, and C. R. Kennedy. 1997. A checklist of metazoan parasites of cichlid fish from Mexico. Journal of the Helminthological Society of Washington 64:195–207.

Scholz, T., J. Vargas-Vázquez, F. Moravec, C. Vivas-Rodríguez, and E. Mendoza-Franco. 1995a. Cenotes (sinkholes) of the Yucatan Peninsula, Mexico, as a habitat of adult trematodes of fish. Folia Parasitologica 42:37–47.

———, ———, ———, ———, **and** ———. 1995b. Metacercariae of trematodes of fishes from cenotes (=sinkholes) of the Yucatan Peninsula, Mexico. Folia Parasitologica 42:173–192.

———, ———, ———, ———, **and** ———. 1996. Cestoda and Acanthocephala of fish from cenotes (sinkholes) of the Peninsula of Yucatan, Mexico. Folia Parasitologica 43:141–152.

J. Helminthol. Soc. Wash.
65(1), 1998 pp. 95–98

Research Note

Effect of *Echinostoma caproni* Infection on Survival, Growth, and Fecundity of Juvenile *Biomphalaria glabrata*

KATHARINE A. SCHMIDT,[1] BERNARD FRIED,[1,3] AND M. A. HASEEB[2]

[1] Department of Biology, Lafayette College, Easton, Pennsylvania 18042 and
[2] Department of Microbiology and Immunology, State University of New York, Health Science Center, Brooklyn, New York 11203

ABSTRACT: The effect of *Echinostoma caproni* infection on survival, growth, and fecundity of juvenile *Biomphalaria glabrata* was studied. Of 40 juvenile snails (4 ± 0.2 mm in shell diameter) exposed to 5 miracidia each, 24 were alive at 6-wk postexposure (PE) and 16 of these were infected with *E. caproni* larvae. Of 40 size-matched control snails maintained identically to the experimental snails except for mira-

[3] Corresponding author
(e-mail: friedb@lafvax.lafayette.edu).

cidial exposure, 35 were alive at the end of the 6-wk observation period. The significantly greater survival rate of the control snails suggested that parasitism with larval *E. caproni* adversely affected snail survival. Exposure to *E. caproni* miracidia did not alter the growth of juvenile *B. glabrata* since there was no significant difference in shell diameter of exposed and control snails during the 6-wk observation period. The number of snail egg masses among the unexposed snails was 3.5 to 5.5 times greater than that of exposed snails, suggesting that infection with *E. caproni* adversely affected fecundity in *B. glabrata*.

KEY WORDS: *Echinostoma caproni*, Trematoda, *Biomphalaria glabrata*, Gastropoda, invertebrate–parasite relationships.

Effects of larval trematode infections on their first intermediate host snails have been studied in many host–parasite systems (see Thompson [1985, 1997] and Hurd [1990] for review). However, schistosome–snail interactions have been studied more extensively because of the medical significance of schistosomes. The effects of larval schistosomes on the dynamics of snail populations, as defined by snail survival, growth, and fecundity, have been investigated. For example, Crews and Yoshino (1989) showed that *Schistosoma mansoni* infection reduced the fecundity of *Biomphalaria glabrata*; Mueleman (1972) demonstrated that infection of sexually mature *B. pfeifferi* with *S. mansoni* miracidia resulted in decreased fecundity and accelerated growth. In contrast, Raymond and Probert (1993) showed a reduction of growth of both immature and mature *Bulinus natalensis* infected with *S. margrebowiei*.

Effect of echinostome infection on survival, growth, and fecundity of snail hosts has received little attention. Christensen et al. (1980) noted that various species of *Biomphalaria* infected with miracidia of an Egyptian strain of *Echinostoma caproni* (referred to as *E. liei* in their study) showed an increased death rate compared with the uninfected control snails. Details of their experiments were not given. Kuris (1980) reported that infection with an Ethiopian strain of *E. caproni* (referred to as *E. liei* in his study) decreased the growth rate and increased the mortality of 1–2-mm juvenile *B. glabrata*, but had no effect on growth and survival of 4–6-mm juvenile snails.

The *E. caproni–B. glabrata* model has been relatively unexplored for studies on larval trematode–gastropod relationships. This model is particularly useful because this echinostome can be cycled easily in the laboratory between *B. glabrata* snails and mice, chicks or hamsters (see Fried and Huffman [1996] for review). The purpose of the present study was to determine the effect of infection with an Egyptian strain of *E. caproni* on survival, growth, and fecundity of juvenile *B. glabrata* (4 ± 0.2 mm in shell diameter).

In each of 4 experiments, 10 *B. glabrata* snails (4 ± 0.2 mm in shell diameter) were exposed individually to 5 miracidia of *E. caproni* in 1.5 ml of artificial spring water (ASW; Ulmer, 1970) in a multiwell chamber for 24 hr, and an identical population of snails was treated the same way but not exposed to miracidia. Experimental and control snails were placed in separate 400-ml plastic beakers, 10 per 350 ml of ASW.

Snails were fed boiled leaf lettuce ad libitum supplemented with occasional feeding of TetraMin (Tetra Werka, Melle, Germany), maintained at 23 ± 1°C, and the water was changed twice weekly. Snail shell diameters were measured weekly for 6 wk of postexposure (PE). The number of live snails, number of egg masses, and number of eggs per mass were recorded weekly. At 6-wk PE, the exposed snails were necropsied to determine the presence of rediae in the digestive gland–gonad complex (DGG).

Survival and growth data between exposed and control groups were compared using a two-tailed Kruskal–Wallis analysis of variance test (True Epistat™, Epistat Services, Richardson, Texas), with $P < 0.05$ considered significant.

Of the 40 exposed and 40 control snails used, 24 and 35 snails, respectively, were alive at 6-wk PE. Of the 24 exposed snails examined at 6-wk PE, 16 were infected with *E. caproni* rediae. Mean weekly survival of exposed and control snails is shown in Figure 1. The survival of snails exposed to *E. caproni* miracidia decreased significantly during the 6-wk PE ($P = 0.003$).

Increased snail mortality probably resulted from extensive damage to the DGG of infected snails (see Fig. 16 in Fried and Huffman [1996]). Such damage resulted in extensive disruption of the architecture of the DGG. The effects of redial infection with *E. caproni* on organs other than the DGG have not been studied.

Mean shell diameters of exposed and control snails are shown in Figure 2. There was no significant difference in the shell diameter of con-

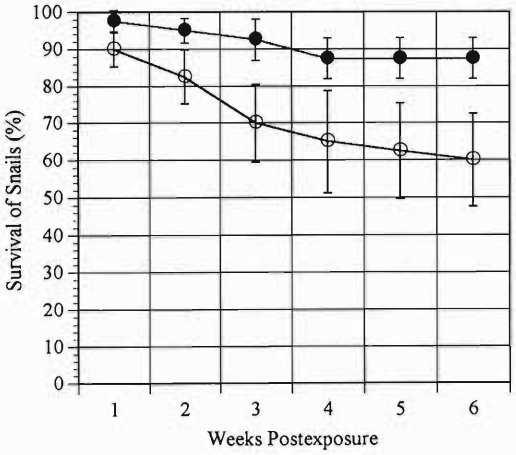

Figure 1. Percent survival of juvenile *Biomphalaria glabrata* with and without exposure to *Echinostoma caproni* miracidia (open circles = exposed snails; closed circles = unexposed snails).

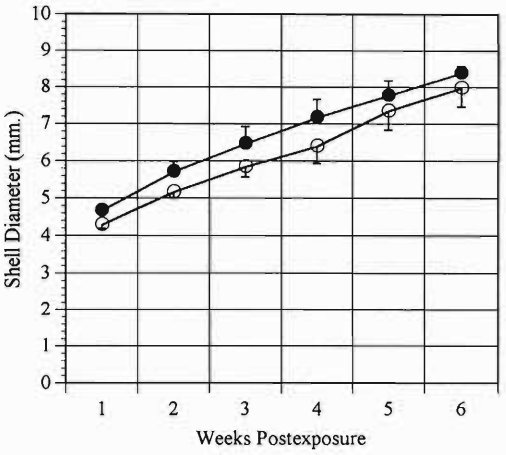

Figure 2. Mean shell diameter of juvenile *Biomphalaria glabrata* with and without exposure to *Echinostoma caproni* miracidia (open circles = exposed snails; closed circles = unexposed snails).

trol snails versus those exposed to *E. caproni* during the 6-wk PE ($P = 0.851$).

Egg masses were first seen in 2 control groups and 1 exposed group at 5-wk PE. All control groups had egg masses at 6-wk PE, while only 2 of 4 of the experimental groups had egg masses at this time. Egg masses from infected snails were often misshapen and contained fewer embryos per egg mass than those from uninfected snails. The mean ± SEM number of egg masses per snail in the control groups at weeks 5 and 6 PE was 0.4 ± 0.2 and 0.3 ± 0.1, respectively. The mean ± SEM number of egg masses per snail in the exposed groups at weeks 5 and 6 PE was 0.1 ± 0.1. The mean ± SEM number of eggs per snail in the control groups at weeks 5 and 6 PE was 3.3 ± 1.9 and 3.2 ± 1.0, respectively. The mean ± SEM number of eggs per snail in the exposed groups at weeks 5 and 6 PE was 0.6 ± 0.6 and 0.9 ± 0.5, respectively. The mean number of eggs in control groups was 5.5 times greater than in experimental groups at 5-wk PE and 3.5 times greater at 6-wk PE. The number of eggs in the experimental groups was significantly less ($P < 0.05$) than that of the control groups at 5 and 6 weeks PE.

Survival of the exposed snails was significantly reduced compared to that of the unexposed snails ($P = 0.003$), suggesting that larval parasitism by the Egyptian strain of *Echinostoma caproni* adversely affected the survival of juvenile *Biomphalaria glabrata.* Our finding supports the previous observation by Christensen et al. (1980) on increased mortality of *Biomphalaria* snails infected with an Egyptian strain of *E. caproni.* However, our finding differs from that of Kuris (1980), in which exposure to an Ethiopian strain of *E. caproni* had no effect on the survival of 4–6-mm *B. glabrata.* Factors other than parasite strain differences may account in part for discrepancies seen in the 2 studies. Thus, whereas we infected individual snails each with 5 miracidia, Kuris exposed 25 snails en masse to 125 miracidia; temperature of snail maintenance was not given in that study, whereas we maintained our snails at 23 ± 1°C.

Our results suggest that larval parasitism of *E. caproni* in *B. glabrata* did not alter snail growth, at least based on maximal shell diameters of the snails. Moreover, there is no evidence for gigantism in this model as reported for various snail–larval trematode systems (see Thompson [1985, 1997] and Hurd [1990] for review). Kuris (1980) noted a decline in growth of 1–2-mm *B. glabrata* infected with the Ethiopian strain of *E. caproni,* but no adverse effect of parasitism on the growth of snails exposed at 4–6 mm was recorded.

Fecundity of snails was not studied by Kuris (1980), but results of our studies show that the number of eggs laid by control snails was 3.5 to 5.5 times greater than that laid by the exposed snails at 5- to 6-wk PE. In accord with numerous studies on larval trematode infections in snails,

juvenile *B. glabrata* infected with *E. caproni* larvae showed reduced fecundity.

Literature Cited

Christensen, N. Ø., F. Frandsen, and M. Z. Roushdy. 1980. The influence of environmental conditions and parasite–intermediate host-related factors on the transmission of *Echinostoma liei*. Zeitschrift für Parasitenkunde 63:47–63.

Crews, A. P., and T. P. Yoshino. 1989. *Schistosoma mansoni*: effect of infection on reproduction and gonadal growth in *Biomphalaria glabrata*. Experimental Parasitology 68:326–334.

Fried, B., and J. E. Huffman. 1996. The biology of the intestinal trematode *Echinostoma caproni*. Advances in Parasitology 38:311–368.

Hurd, H. 1990. Physiological and behavioural interactions between parasites and invertebrate hosts. Advances in Parasitology 29:272–317.

Kuris, A. M. 1980. Effect of exposure to *Echinostoma liei* miracidia on growth and survival of young *Biomphalaria glabrata* snails. International Journal for Parasitology 10:303–308.

Mueleman, E. A. 1972. Host–parasite interrelationships between the freshwater pulmonate *Biomphalaria pfeifferi* and the trematode *Schistosoma mansoni*. Netherlands Journal of Zoology 22:355–427.

Raymond, K., and A. J. Probert. 1993. The effect of infection with *Schistosoma margrebowiei* on the growth of *Bulinus natalensis*. Journal of Helminthology 67:10–16.

Thompson, S. N. 1985. Review: metabolic integration during the host associations of multicellular animal endoparasites. Comparative Biochemistry and Physiology 81B:21–42.

———. 1997. Physiology and biochemistry of snail larval trematode relationships. Pages 149–195 *in* B. Fried and T. K. Graczyk, eds. Advances in Trematode Biology. CRC, Boca Raton, Florida.

Ulmer, M. J. 1970. Notes on rearing of snails in the laboratory. Pages 143–144 *in* A. J. MacInnis and M. Voge, eds. Experiments and Techniques in Parasitology. W. H. Freeman, San Francisco.

J. Helminthol. Soc. Wash.
65(1), 1998 pp. 98–102

Research Note

Helminth Parasites of the Spotted Salamander *Ambystoma maculatum* and Red-backed Salamander *Plethodon c. cinereus* from Northwestern Wisconsin

MATTHEW G. BOLEK AND JAMES R. COGGINS

Department of Biological Sciences, University of Wisconsin–Milwaukee, Milwaukee, Wisconsin 53201 (e-mail: bolek@csd.uwm.edu, coggins@csd.uwm.edu)

ABSTRACT: Twenty spotted salamanders *Ambystoma maculatum* and 20 red-backed salamanders *Plethodon c. cinereus* were collected from NW Wisconsin in May 1996 and examined for helminth parasites. Two species of helminths infected the spotted salamanders, while 3 species infected the red-backed salamanders. The nematode *Batracholandros magnavulvaris* had the higest prevalence in spotted salamanders (45%), while the nematode *Rhabdias* sp. had the highest prevalence in red-backed salamanders (30%). The trematode *Brachycoelium salamandrae* had the highest mean intensity in both hosts, 3.3 in *A. maculatum* and 2.0 in *P. c. cinereus*. This is the first report of *B. magnavulvaris* from *Ambystoma maculatum* as well as the first report of it from Wisconsin.

KEY WORDS: *Ambystoma maculatum, Plethodon c. cinereus, Brachycoelium salamandrae, Batracholandros magnavulvaris, Rhabdias* sp., Wisconsin.

The spotted salamander *Ambystoma maculatum* Shaw, 1802, is a large, robust species of mole salamander reported from south-central Ontario to Nova Scotia, south to Georgia and eastern Texas (Vogt, 1981). The red-backed salamander *Plethodon c. cinereus* Green, 1818, is one of the smallest woodland species of lungless salamanders that occurs throughout the northeastern United States and southeastern Canada, with populations in Ontario, Minnesota, Missouri, Arkansas, Oklahoma, Louisiana, and Georgia (Vogt, 1981). Both species inhabit mesic forests throughout northern Wisconsin. Although parasites of the spotted salamander, *Ambystoma maculatum*, and the red-backed salamander, *Plethodon c. cinereus*, have been studied by several